

**P1 Event-Related Potential Component Modulations and Behavioural Inhibitory  
Cueing Effects in the Presence of a Distractor Stimulus**

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## Statement of Sources

I declare that this report is my own original work and that contributions of others  
have been duly acknowledged.

Date:

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## Table of Contents

Abstract.....	1
Introduction.....	2
1.2 Two Separate Mechanisms of ICE .....	5
1.2.1 Output ICEs .....	5
1.2.2 Input ICEs .....	6
1.2.3 Input and Output ICE Dissociations .....	8
1.2.4 Implications for This Study .....	10
1.3 Electroencephalography (EEG) and Event Related Potentials (ERPs).....	11
1.3.1 The P1 Component .....	12
1.3.2 P1 within ICEs .....	12
1.4 The Distractor Stimulus .....	14
1.5 Attention Deficit/Hyperactivity Disorder .....	15
1.6 Study Aims and Hypotheses .....	16
Method.....	18
2.2 Participants.....	18
2.3 Design and Analysis .....	18
2.4 Apparatus and Materials .....	19
2.5 Procedure .....	21
Results.....	24

3.2 Behavioural Reaction Times .....	24
3.3 P1 Amplitudes.....	25
3.3.1 Ipsilateral electrodes .....	25
3.3.2 Contralateral electrodes .....	29
3.4 Correlational Analyses .....	32
Discussion .....	34
4.2 Behavioural Reaction Time .....	34
4.3 P1 Amplitudes.....	36
4.4 P1 cueing effect by behavioural ICE Correlations .....	38
4.5 Limitations .....	38
4.6 Future Research .....	40
4.7 Summary and Conclusion .....	41
References.....	43

## **List of Tables and Figures**

<i>Figure 1.</i> Time course of the spatial cueing task employed .....	20
<i>Table 1.</i> Behavioural Reaction Times Descriptives .....	25
<i>Table 2.</i> Ipsilateral P1 amplitude Descriptives .....	27
<i>Figure 2.</i> Control: Ipsilateral electrode grand mean waveforms .....	28
<i>Figure 3.</i> Deficit: Ipsilateral electrode grand mean waveforms for cued and uncued trials....	28
<i>Table 3.</i> Contralateral P1 amplitude Descriptives .....	30
<i>Figure 4.</i> Contralateral electrode grand mean waveforms.....	31
<i>Figure 5.</i> Contralateral electrode grand mean waveforms.....	31
<i>Table 4.</i> Oculomotor Activated Correlation Table .....	32
<i>Table 5.</i> Oculomotor Suppressed Correlation Table.....	33

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## **Abstract**

Inhibitory cueing effects (ICEs) denote slowed responses to a target stimulus caused by exposure to a cue appearing in the same location and are thought to improve the efficiency of visual search. Research has demonstrated the existence of two types of ICE – those that are generated along input pathways (sensory/perceptual; observed when the oculomotor system is suppressed) and those that are generated along output pathways (oculomotor; observed when the oculomotor system is active). Within a spatial cueing task using oculomotor suppressed (for input ICEs) and oculomotor active (for output ICEs) manipulations, the present study employed electroencephalography to study the effects of input and output ICEs on an early sensory/attentional event-related potential component (the P1) in the presence of a distractor stimulus. This study also explored the effects of ADHD symptomology on ICEs. Results showed ICEs (slower reaction times for cued trials compared to uncued trials) for both suppressed and active manipulations, but no difference in the magnitude of ICEs between the two. Additionally, while there was no overall difference in RTs between deficit and control levels, there was a marginally significant interaction such that controls had a significant ICE, but deficits' ICEs were marginal. No significant results were observed for P1 analyses. Results, limitations and future directions are discussed.



Visual search is a fundamental part of everyday life (Eckstein, 2011). Consider one trip to the supermarket: for each item that we require we must search for the correct aisle, and within that aisle the correct shelf, and within that shelf the correct item. Every item will require many eye movements and shifts of attention as one scans the myriad items to find the right one. Visual search also has a survival element. This would include the ability of a pedestrian to scan a road for cars before crossing it, and prehistoric peoples scanning the savannah for predators and prey or foraging for food (Klein & McInnes, 1999). Thus, mechanisms for increasing the efficiency of visual search are paramount for functionally navigating and surviving in one's environment.

The efficiency of visual search has been found to rely on interactions between the facilitation of orienting attention toward salient stimuli and the inhibition of orienting attention away from irrelevant stimuli (Luck & Hillyard, 1994). The orienting of visual attention refers to the aligning of visual attentional resources with a certain space or object (Posner, 1980). One such proposed orienting mechanism for increasing the efficiency of visual search is known as inhibition of return (IOR; Posner & Cohen, 1984). IOR refers to an inhibited response to spaces or objects that have been previously oriented (Posner & Cohen, 1984). By placing inhibitory 'tags' on previously oriented spaces or objects, IOR is thought to facilitate searching of unexplored spaces or objects, thus preferencing novelty (Klein, 1988; Klein & McInnes, 1999). This phenomenon has been most extensively studied in variations of Posner's (1980) spatial cueing task (Chica, Arévalo, Botta, & Lupiáñez, 2014).

Spatial cueing tasks have a simple design. Typically, a participant responds as fast as they can to a target appearing to the left or right of a centre fixation point. Targets appear after the presentation of a cue that appears at, or points to, the subsequent location of the target (cued trials) or a different location (uncued trials; Posner & Cohen, 1984). Within spatial cueing tasks, the general pattern of cueing effects has two phases: when the time

interval between the presentation of a cue and then a target (cue-target onset asynchrony; CTOA) is short ( $< 300$  ms), responses show faster reaction times (RTs) to cued targets than to uncued targets (facilitation; Samuel & Kat, 2003). However, at longer CTOAs (300ms to 3000ms) responses to cued targets have slower RTs than those appearing in an uncued space (inhibition; Samuel & Kat, 2003). In line with the present study's aims, only the inhibitory phase will be discussed here.

Of fundamental importance to the observation of this inhibition is that the cues are non-predictive of the location of the target (Berlucchi, 2006). That is, cues appear on the same side as the target only 50% of the time. If cues reliably predict the location of the target, facilitation to cued targets is generally observed at most CTOAs as such cues encourage strategic anticipation of the target's appearance (Summerfield & Egner, 2009). Most IOR studies employ cues and targets that appear in one of two possible locations (i.e., peripheral cues and targets; Chica et al., 2014). Peripheral cues and targets reflexively orient attention to a space through sensory stimulation (Lim, Eng, Janssen, & Satel, 2018). IOR has also been found using arrows that appear at fixation and point to a location (central arrow cues and targets, Rafal, Calabresi, Brennan, & Sciolto, 1989). Central arrows are thought to induce endogenous orienting as the arrows' meaning must be inferred before attention shifts (Gabay, Avni, & Henik, 2002). In addition to cue-target combinations, inhibition has been observed using different response modalities (saccades, manual button push) to both cues and targets (Klein, 2000). Moreover, the elicitation of inhibition differs depending on what combination of cues, targets and response modalities are employed. For example, any cue-target combinations that employ a saccade (a quick movement of the eye to align the fovea with a stimulus) at any point during a trial tend to generate inhibition (Taylor & Klein, 2000). However, when saccadic responses are suppressed (eyes remain fixated for the trial duration) inhibition is observed only when peripheral cues are used (Taylor & Klein, 2000).

This has led many researchers to conclude that there exists two separate inhibitory cueing mechanisms (discussed further in Section 1.2): (1) inhibition that is generated along input pathways (sensory/perceptual) that can be observed when the oculomotor system is suppressed (i.e., eyes remain fixated) and thought to be a short lasting effect, and (2) a longer lasting inhibition that is generated along output pathways that can be observed when the oculomotor system is activated (i.e., a saccade is required to the cue or target; Hilchey, Klein, & Satel, 2014). Some researchers have argued that only inhibition in output pathways can be considered ‘true’ IOR as a mechanism for orienting during visual search would need to be closely related to oculomotor activation (Hilchey et al., 2014). Other researchers have argued that in fact all observed inhibition is input based (Fecteau & Munoz 2005). That is, inhibition is caused at a sensory or perceptual level and, as a result, delays processing all the way through to the output stage. Indeed, sensory, perceptual, attentional and motoric factors have all been shown to elicit inhibition on cued trials (Berlucchi, 2006). Thus, to stay theoretically neutral, the current study will employ the term “inhibitory cueing effect” (ICE) to denote any slowed RTs due to the appearance of a cue in the same location as a target (Hilchey et al., 2014). Observed inhibition that is thought to be generated closer to input pathways will be referred to as input ICEs and those thought to be generated closer to output pathways will be referred to as output ICEs.

While behavioural data on ICEs is well established, researchers are yet to find a reliable electrophysiological marker of ICEs. The aim of the present study is to further explore the nature of input and output ICEs by studying the relationship between a potential electrophysiological marker (the P1 event-related potential component) and input and output ICEs.

## 1.2 Two Separate Mechanisms of ICE

### 1.2.1 Output ICEs

It has been proposed that output ICEs are generated following the priming of oculomotor circuitry that occurs as a result of preparation or execution of a saccade to a certain space. After initial facilitation, programmed eye-movements become inhibited to prevent reorienting and thus a slowed response is observed at the previously oriented space (Klein & Taylor, 1994).

One line of evidence for the involvement of the oculomotor system comes from Rafal et al. (1989) who gave their participants one of three instructions in relation to how they should respond to a central arrow cue: (1) execute a saccade to the cue, (2) plan a saccade to the cue (without execution), or (3) stay fixated (with no further instruction). ICEs were observed in equal magnitude for conditions that required participants to either execute or plan a saccade. However, when participants were instructed to simply stay fixated, no ICE was observed. It is important to emphasise that the observed ICEs for planned and executed saccades were of equal magnitude. This indicates that executing a saccade has no added effect beyond that of simply preparing to make a saccade and thus, likely to be caused by the same mechanism. Additionally, the use of central arrows was important, as it implies that there was no peripheral stimulation causing reflexive orienting which indicates that orienting most likely occurred because of planning or execution of a saccade. In conjunction with observing no ICE when participants were instructed to simply stay fixated, this implicates the priming of oculomotor pathways in generating ICEs (Rafal et al., 1989).

Another line of evidence for output ICEs comes from converging evidence implicating the superior colliculus (SC) in generating ICEs. The SC is a midbrain structure responsible for the programming and initiation of saccades (Munoz, 2002). Three lines of evidence for the involvement of the SC in generating ICEs are: (1) ICEs are greatly

diminished in patients with a degenerated SC (Posner, Rafal, Choate & Vaughn, 1985), (2) using monocular vision, ICEs are more pronounced (uncued minus cued RT difference is larger) in the temporal hemiretina compared to the nasal hemiretina (Rafal et al., 1989). If the SC is involved in generating ICEs, this pattern of results is expected because the temporal hemiretina is where the SC gets most of its input (Sapir, Soroker, Berger, & Henik, 1999). (3) Single cell recordings of rhesus monkeys performing a spatial cueing task have shown moderate correlations between motor neurons in the intermediate layers of the SC (iSC) and behavioural ICEs (Fecteau & Munoz, 2005). Taken together, this evidence implicates the iSC in generating ICEs and, by extension, because the iSC initiates saccades, it is evidence of the oculomotor systems involvement in generating ICEs.

### **1.2.2 Input ICEs**

Input ICEs are any ICEs that originate closer to the input stage of processing and are observed when the oculomotor system is suppressed. The most investigated source of input ICEs is sensory adaptation (Dukewich, 2009; Hilchey et al., 2014; Satel, Hilchey, Wang, Story, & Klein, 2013). Sensory adaptation can be defined as a reduction in the response of neurons due to neuronal fatigue from repeated exposure to stimuli (Kohn, 2007). When peripheral cues and targets are used, cued trials cause repeated peripheral stimulation (RPS) because the cue appears in the same location as the target in the participants peripheral vision and therefore stimulates the same neurons in early visual areas (Dukewich & Boehnke, 2008). In contrast, uncued trials do not experience RPS because the cue and target appear at different locations. It has been proposed that RPS on cued trials is causing sensory adaptation that is not experienced on uncued trials (Dukewich & Boehnke, 2008). Therefore, because of RPS, sensory adaptation causes slowed responses to cued trials in comparison to uncued trials.

Dukewich and Boehnke (2008) tested this theory by exposing participants to 1-5 peripheral cues presented sequentially before responding to a target. They demonstrated that on cued trials responses became increasingly slower with the addition of each cue. In contrast, the uncued trial RTs were both faster than cued trials and remained relatively constant no matter how many cues were presented. This indicates that the repetition of the cue in cued trials is playing a large role in slowing responses and thus provides evidence for the role of sensory adaptation in causing ICEs.

A second line of evidence for input ICEs comes from single cell recordings in the superficial layers of the SC (or input areas of the SC; sSC) in rhesus monkeys while they performed a spatial cueing task using non-predictive cues (Dorris, Klein, Everling, & Munoz, 2002; Fecteau & Munoz, 2005). While successfully inducing ICEs, these studies found reduced visual neuron activity in the sSC on cued trials compared to uncued trials. Moreover, the uncued-cued difference in sSC activity grew in magnitude as the CTOA increased (up to 500 ms). Visual neurons in the sSC respond to sensory input but they are not implicated in generating saccades (Rodieck & Watanabe, 1993; Fries, 1984; Perry, Oehler & Cowey, 1984), thus indicating that the reduced activity is more likely due to sensory adaptation. Since the neuroanatomy of other primate's visual systems is very similar to humans, the neuroactivity associated with ICEs is likely to be analogous to that of humans (Munoz, 2002).

Based on such findings, it has been argued that in fact all ICEs are generated in input pathways whose observed effects carry all the way down the processing chain (Fecteau & Munoz, 2005). One limitation of such an assertion is that these sensory effects were observed at relatively short CTOAs (<610 ms; Dorris, Klein, Everling, & Munoz, 2002; Dukewich & Boehnke, 2008; Fecteau & Munoz, 2005) while the behavioural effects were still observed after these sensory effects had diminished. Additionally, in human subject studies, ICEs have

been observed at much longer CTOAs ( $> 1000$  ms; Hilchey et al., 2014; Satel et al, 2013), indicating the possibility of multiple mechanisms of inhibition.

The following section will discuss evidence showing dissociations between oculomotor activated and oculomotor suppressed conditions and how this raises the possibility that there exists two discrete mechanisms.

### **1.2.3 Input and Output ICE Dissociations**

So far, it has been shown that oculomotor programming is implicated in generating ICEs (output pathways; Rafal et al., 1989; Posner et al. 1985) and sensory adaptation can also elicit ICEs (input pathways; Dukewich & Boehnke, 2008). But, are these effects a reflection of one continuous phenomenon, or (at least) two separate ICE causing phenomena? Studies have shown that the two can be dissociated from one another.

Perhaps the most comprehensive test of this question comes from Taylor and Klein (2000) who found evidence for two separate (input and output) types of ICE. They manipulated cue type (peripheral or central arrow), target type (peripheral or central arrow), cue response modality (ignore, saccade, or manual button push) and target response modality (saccade or manual button push). By using every possible combination of cue, target, and response type this allowed them to directly compare the conditions under which ICEs are both generated (at cue onset) and subsequently observed (at target onset). Their findings indicated a dissociation between input and output ICEs. In almost all conditions when a saccade was required, an ICE was observed. This occurred for both peripheral and central cues and targets. Importantly, the magnitude of IOR was the same whether cues or targets were peripheral or central arrows. That is, because only peripheral cues and targets induce reflexive orienting and RPS, the observation that central arrow cues and targets produce the same sized effect implies it is due to oculomotor inhibition, not sensory level effects.

Conversely, in conditions that did not use saccades, ICEs were only observed in response to peripheral targets. Again, because only peripheral stimuli cause reflexive orienting through peripheral stimulation, this result indicates a sensory, or input-based ICE, distinct from those observed with saccades. These results were replicated by Hilchey et al. (2014). Importantly for the present study, they found ICEs at a 1050ms CTOA with the oculomotor system suppressed (no-eye movement). This is beyond the amount of time that is expected for neurons to recover to normal functioning after sensory adaptation (Fecteau & Munoz, 2005) suggesting there may be other input ICEs occurring at long CTOAs.

Dissociations have also been found employing short-wave light frequencies (s-cone frequencies) whose projections from the retina bypass the SC (Sumner, Nachev, Vora, Husain, & Kennard, 2004). Sumner et al. (2004) compared s-cone frequency cues against normal luminance cues (both peripheral) while employing both oculomotor activated and suppressed conditions. This produced two distinct patterns. Firstly, in the suppressed condition (manual button response to target) ICEs were observed for both normal luminance and s-cone frequencies. However, in the activated condition (saccade to target), an ICE was found with normal luminance, but not s-cone frequencies. This finding indicates that ICEs can be induced without mediation from the SC (because s-cone frequencies bypass it), but only when the oculomotor system is suppressed. This is a noteworthy finding as evidence for input and output ICEs implicate the SC – for input ICEs the sSC, and for output ICEs the iSC.

An alternative explanation for ICEs in general was proposed by Dukewich (2009). She proposed that, through non-associative learning, responses to cued targets become habituated and thus, reduce neuronal responsiveness. That is, through repeated exposure to non-predictive cues, cues are learnt to be irrelevant to achieving the goal (i.e., locating and responding to the target) and thus, less attentional resource is allocated to them which slows response times to cued targets.



### **1.2.4 Implications for This Study**

Taken together, the evidence presented in this section indicates that ICEs are differentially elicited depending on the activation status of the oculomotor system. Thus, to effectively study ICEs under oculomotor activated or suppressed conditions, careful manipulation and monitoring of eye movements is required. Investigating input ICEs requires a condition that successfully suppresses the reflexive saccades generated by peripheral cues to ensure that oculomotor activation is not contributing to the effect (Hilchey et al., 2014). Conversely, to investigate output ICEs requires a condition that ensures a saccade is executed at the right time and in the right location (Hilchey et al., 2014). Many past studies (Taylor & Klein, 2000; Sumner et al., 2004; Rafal et al., 1989) gave explicit instructions to make a saccade or stay fixated, however saccades were not actively monitored to ensure the instruction was followed correctly. To allay this concern, more recent studies have employed eye tracking hardware to closely monitor saccades (Hilchey et al. 2014; Lim et al., 2018; Satel Wang, Hilchey, & Klein, 2012). If an incorrect saccade is made, the participant is given an explicit message to inform them of such and that trial is either discarded or recycled. This ensures that every trial that is analysed is being conducted correctly. For these reasons, eye tracking will be employed in this study.

Another implication of these findings is that, if oculomotor suppression and activation generate different forms of ICE they should be represented by different electrophysiological markers. However, while behavioural data on ICEs is well established, a reliable electrophysiological marker of ICEs is yet to be found. The following section discusses evidence for one extensively explored electrophysiological marker: the early sensory P1 ERP component.

### 1.3 Electroencephalography (EEG) and Event Related Potentials (ERPs)

An EEG system is a brain imaging apparatus that uses electrodes to measure electrical voltage across the scalp. EEG measures voltage polarity (positive or negative), amplitude, and the temporal length of voltage deflections that reflect post-synaptic activity in cortical pyramidal neurons (Kirschstein & Kohling, 2009). Voltage deflections that reliably occur in response to an observed event are known as event related potentials (ERPs). However, there is a lot of noise (irrelevant signal) in an EEG readout which can often mask the neural activity of interest. To circumvent this problem EEG is time-locked to the beginning of an experimental event (e.g., target onset) and the event is repeated several hundred times for each participant. Repeated trials are then averaged together to improve the signal to noise ratio and thus, reveal the underlying ERP (Luck & Kappenman, 2012). Each separate deflection in an ERP represents a ‘component’ of that experimental event and are thought to represent separate processes in the underlying neural structures (Luck & Kappenman, 2012).

Due to the near instantaneous movement of electricity, ERPs have excellent temporal resolution. That is, ERPs are very good at measuring *when* an event occurs. Thus, ERP analyses have been used extensively to study phenomena that changes rapidly from moment to moment, such as ICEs (e.g., Hopfinger & Mangun, 1998; Prime & Ward, 2005, 2006; Satel et al., 2013). The present study will investigate the relationship between modulations of the P1 component and behavioural ICEs. Therefore, it is important to first discuss the P1 component and what it is thought to represent.

### **1.3.1 The P1 Component**

The P1 ERP component (henceforth referred to as simply, P1) is the first positive fluctuation in an ERP, peaking approximately 100ms after the time locked onset of an experimental event. Localisation studies have shown that in the visual system, P1 probably originates within the lateral occipital complex (Di Russo, Martinez, & Hillyard, 2003). P1 deflections can be observed and modulated in response to sensory input and basic physical characteristics such as luminance, regardless of any higher order attentional processes (Johannes, Miinte, Heinze, & Mangun, 1995). Beyond this, P1 has been proposed to represent part of a ‘sensory gain control’ mechanism (Hillyard, Vogel, & Luck, 1998). It is proposed that sensory gain control improves the signal to noise ratio of incoming signals by amplifying attended stimuli and suppressing unattended stimuli. The P1 is thought to index the “suppressor” in the sensory gain control mechanism (Hillyard et al., 1998; Luck & Hillyard, 1994). That is, when a space or object is oriented, surrounding irrelevant stimuli’s signal will be actively suppressed, reflected in strong P1 amplitudes. However, if stimuli appear in unoriented space this suppression reduces to allow for reorienting, reflected in decreased P1 amplitudes (Hillyard et al., 1998; Luck & Hillyard, 1994).

### **1.3.2 P1 within ICEs**

Most studies of ICEs that have employed EEG have observed a “P1 cueing effect” (Hopfinger & Mangun 1998; Prime & Ward, 2005, 2006; Satel et al., 2013; Satel et al., 2012). The P1 cueing effect is a reduction in P1 amplitude for cued trials compared to uncued trials. The P1 cueing effect has been largely attributed to modulations at a sensory level from phenomena such as refraction and sensory adaptation (Satel et al., 2013; Satel et al., 2012) due to RPS on cued trials. One line of evidence for this comes from Satel, et al. (2012) who found a P1 cueing effect in retinotopically cued locations (locations corresponding to inputs

in the retina), but not spatiotopically cued locations (locations corresponding to the environment). Retinotopic coordinates are coded at input locations (i.e., the sSC) and then transferred to higher cortical areas where they are remapped spatiotopically (Turi & Burr, 2012). Thus, the observation of the P1 cueing effect only in retinotopic locations indicates it is most closely linked with input ICEs.

Of most importance to this study, Satel et al. (2013) produced a P1 cueing effect in oculomotor suppressed and activated conditions at a long CTOA (1200 ms). However, only the suppressed condition revealed a moderate negative correlation ( $r = -.38$ ) between P1 cueing effects and observed ICEs while the activated condition had no such correlation. This correlation was stronger ( $r = -.60$ ) using meta-analytic data from 19 other studies employing suppressed conditions. The correlation demonstrated that as observed ICEs became larger in the suppressed condition, so too did the P1 cueing effect. This indicates that the P1 cueing effect is more closely related to input ICEs. The observation of a P1 cueing effect with no correlation in the activated condition indicates that input ICEs are still being generated while making a saccade, but their observed effect is being masked by the output ICE effect. This is supported by behavioural RTs that show significantly greater inhibition in the saccade condition, suggesting the presence of an extra inhibitory mechanism when the oculomotor system is activated compared to when it is suppressed. Further support for P1 cueing effects representing input ICEs came in a follow-up study (Satel, Hilchey, Wang, Reiss, & Klein, 2014) that employed peripheral and central arrow cues which activated the oculomotor system. This study observed ICEs behaviourally in both conditions, but a P1 cueing effect *only* when using peripheral cues, not with central arrow cues. If the P1 cueing effect represents sensory adaptation through RPS, this result would be expected as only peripheral cues cause RPS.

However, consistent with Hilchey et al.'s (2014) observation of an ICE in a suppressed condition at long CTOAs (discussed in Section 1.2.3), Satel et al.'s (2013) observed P1 cueing effect was also observed at a long CTOA (after sensory adaptation is thought to have subsided). This result is expected when the oculomotor system is activated as output-based ICEs are longer lasting (Lim et al., 2018). However, this result is more difficult to explain for input ICEs. It is possible this evidence indicates that the P1 modulation and behavioural ICEs may represent something other than sensory adaptation at long CTOAs. For example, consistent with sensory gain control, P1 reductions to cued trials may represent a retinotopic suppression of the cued area after the facilitation period has passed (Luck & Hillyard, 1994; Satel et al., 2012). Alternatively, as discussed earlier Dukewich's (2009) theory of inhibition caused by habituation through non-associative learning (which is thought to be informed by sensory adaptation) maybe causing ICEs at longer CTOAs. However, to explore such possibilities, sensory adaptation must be controlled for. To achieve this, balancing sensory stimulation across visual fields is necessary.

#### **1.4 The Distractor Stimulus**

A distractor stimulus is any stimulus that presents as an alternative to the actual target and thus, forces one to discriminate between the distractor and the target. In spatial cueing tasks, a distractor is a secondary stimulus that appears simultaneously with the target at the opposite location. Distractor stimuli have been used in past studies to balance sensory stimulation between visual fields (McDonald, Hickey, Green, & Whitman, 2009; Yang, Yao, Ding, Qi, & Lei, 2012). Although there is still some sensory imbalance within trials (because the cue still appears by itself), it is thought to minimise sensory imbalance across trials (McDonald et al., 2009). Moreover, the addition of a second stimulus adds a measure of spatial discrimination (distinguishing between multiple stimuli in multiple spaces) that have

also been shown to elicit ICEs at long CTOAs (Eng, Lim, Janssen, & Satel, 2018). Thus, while minimising sensory imbalance and eliciting ICEs in a task that requires spatial discrimination, it may be possible to investigate whether the P1 cueing effect is explained by sensory adaptation or something else (e.g., sensory gain control or habituation; Hillyard et al., 1998; Dukewich, 2009).

### **1.5 Attention Deficit/Hyperactivity Disorder**

The present study was also interested in investigating the effect of Attention Deficit/Hyperactivity Disorder (ADHD) symptomology in adults on the observation of ICEs. ADHD is categorised in the DSM-5 (APA, 2013) as a neurodevelopmental disorder characterised by either, or both, 1) inattention, or 2) hyperactivity and impulsivity (APA, 2013). While ADHD is thought of mainly as a childhood disorder, impairing levels of ADHD symptomology are experienced by an estimated 65% of adults diagnosed as children (Faraone, Biederman, & Mick, 2006). Moreover, of the 2.3% to 4.5% of adults globally with ADHD, it is estimated that more than one third have not been diagnosed (Feifel & MacDonald, 2008; Kooij et al., 2010; McCarthy, Cranswick, Potts, Taylor, & Wong, 2009).

There are many aspects of ADHD that could affect the observation of ICEs differentially for sufferers in comparison to those who are neurodevelopmentally typical. However, to date only two studies (both using child participants) have looked at the effect of ADHD on ICEs (Fillmore, Milich, & Lorch, 2009; Li, Chang, & Lin, 2002). Both studies indicated reductions in ICEs for children with ADHD compared to a control group. In fact, Fillmore et al. (2009) demonstrated a complete absence of ICEs in those with combined (inattentive and hyperactive) ADHD. Deficits observed in ADHD that may contribute to such a reduction are: a general poorer performance (less accuracy and slower RTS) in various attentional paradigms (Cross-Villasana et al., 2015) and, more importantly, inhibitory deficits

in both motor response (Carr, Nigg & Henderson, 2006) and reflexive orienting (Ortega, Lopez, Carrasco, Anllo-Vento, & Aboitiz, 2013). However, no ICE study has examined adults displaying ADHD symptomology. Given the significant minority of adults with undiagnosed impairing ADHD symptomology (Feifel & MacDonald, 2008), it is possible such deficits may be confounding results. Thus, in addition to looking at input and output ICEs in general the present study will add a self-report assessment of adult ADHD symptomology (Kessler et al., 2005) to separate those who report impairing symptomology from those who do not.

### **1.6 Study Aims and Hypotheses**

The present study will investigate P1 amplitude fluctuations of participants as they complete a spatial cueing task using non-predictive peripheral cues and targets. This study will employ an Oculomotor-Status condition with an oculomotor activated level (saccade to cue; Om-A) and an oculomotor suppressed level (eyes remain fixated; Om-S) to investigate output and input-based ICEs, respectively. Saccades to cues rather than targets will be employed to control for noise caused by oculomotor movements. Saccades will be closely monitored using eye-tracking hardware and software.

The present study aims to further explore the findings of Satel et al. (2013) by employing the same design with three important differences: 1) a distractor stimulus will appear simultaneously with the target to control for sensory imbalance, 2) a longer CTOA (1500 ms) will be employed with the assumption that sensory effects will have sufficiently diminished by this stage, 3) a between subjects “Group” variable will be included to separate those with possible potentially confounding ADHD symptomology (deficit) from those who don’t (control). If the same results as Satel et al., (2013) are found with a sensory balancing distractor and a 1500ms CTOA, this would provide evidence that the P1 cueing effect is not

(completely) due to sensory adaptation. However, if in the presence of a distractor, the P1 cueing effect is not observed, this would be evidence in support of the sensory adaptation explanation:

- (1) Behavioural RTs will be slower in cued trials compared to uncued trials for both Om-A and Om-S conditions. However, due to the additional oculomotor-based inhibition in the Om-A condition, it is expected that there will be an interaction between Cueing and Oculomotor-Status such that the magnitude of difference for RTs between cued and uncued trials will be greater for the Om-A level compared to the Om-S level.
- (2) Based on previous research in attentional paradigms, overall, RTs observed in those classified as having a deficit should be slower than those classified as a control. Moreover, due to proposed inhibitory deficits in those classified as having a deficit, there will likely be an interaction between Cueing and Group such that the magnitude of difference for RTs between cued and uncued trials will be decreased for the deficit level compared to the control level across both levels of Oculomotor-Status.
- (3) Consistent with Satel et al. (2013), P1 amplitude will be reduced in cued trials compared to uncued trials for both Om-A and Om-S conditions. This observation should be decreased in the deficit level compared to the control level of Group.
- (4) There will be a negative correlation between P1 cueing effects (uncued-cued) and behavioural ICEs (uncued-cued) for Om-S, and no correlation for Om-A.



## Method

### 2.2 Participants

An a-priori power analysis indicated that at least 29 participants would be required to reach sufficient power to detect a medium effect ( $f = .25$ ) for the current study. An extra 11 participants were recruited to account for possible outliers, technical difficulties, and ineligibility. Thus, altogether, 40 participants aged between 18 and 47 (25 females; *Mean Age* = 25.00, *SD* = 8.07) were recruited. Criteria for eligibility required participants to be at least 18 years old, have normal or corrected to normal vision, and have no pre-existing neurological disorders. Seven participants were removed due to excessive artefacts in the EEG traces resulting in insufficient trials per conditions. This left a total of 33 participants for analysis (20 females; *Mean Age* = 24.82, *SD* = 8.39). Allocation to the two levels of the between subjects variable, “Group”, was determined by scores on the ADHD Self-Report Scale version 1.1 (ASRS-v1.1; Kessler, 2005; expanded upon in Section 2.4). Those with scores below 17 were allocated to the “control” level and those who scored 17 and above were allocated to the “deficit” level. Participants were recruited through SONA and via word of mouth. As compensation, participants were offered either course credit (where applicable) or monetary compensation (\$15/hour).

### 2.3 Design and Analysis

Data was first inspected to ensure the assumptions of ANOVA were met. Any trials whose RT data was 2.5 units of median absolute deviation (MAD) above or below the overall median were considered outliers and removed (10.7% and 2.2%, respectively).<sup>1</sup> Incorrect

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<sup>1</sup> MAD is used here as it is robust measure of statistical dispersion that is less sensitive to outliers than mean and standard deviation (Leys, Ley, Klein, Bernard, & Licata 2013). This is important in data sets with large amounts of variability (which is common with RT data; Ratcliff, 1993). A moderately conservative ( $\pm 2.5$  units of MAD) exclusion criteria is commonly used in ICE research (Hilchey et al., 2014; Lim et al., 2018)

responses to targets (i.e., indicating the target was left when it was actually right) were also removed (1%). This left a total of 82.2% of trials for analysis.

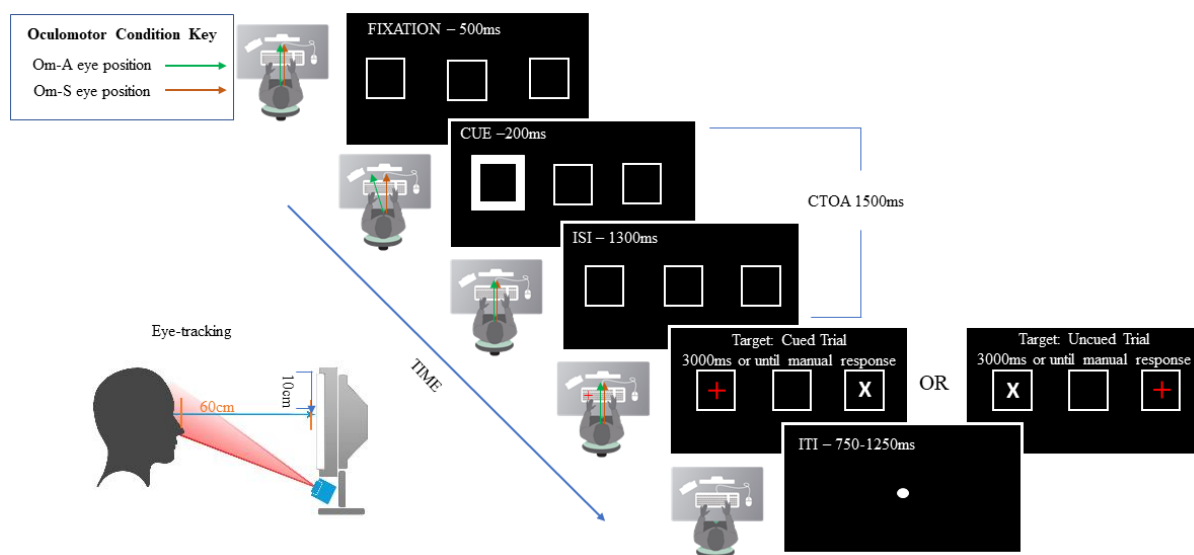
ERP data was extracted using FCz as a reference electrode during acquisition with an offline high pass filter of 1Hz and a low pass filter of 30Hz. All epochs were extracted between -100ms and 400ms around target onset with a baseline correction of 100ms. Any trials with deflections of  $\pm 75$  microvolts were classified as artefacts and removed. P1 data was extracted by locating the most positive point between 100ms and 200ms after target onset with averages taken in a 40ms window around this point.

The design of this study can be described as three 2 (Group: control and deficit) X 2 (Cueing: cued and uncued) X 2 (Oculomotor-Status: Om-A and Om-S) mixed ANOVAs. One ANOVA was used to analyse behavioural RTs (ms) while the other two were used to analyse P1 amplitudes (microvolts or  $\mu V$ ), one for electrodes contralateral to the target and one for electrodes ipsilateral to the target. Post-hoc pairwise comparisons were reported for significant interactions or interactions that were both experimentally relevant and approached significance. Additionally, Pearson's correlations were calculated to investigate the relationship between behavioural ICEs and P1 cueing effects for both ipsilateral and contralateral electrodes across both levels of the Oculomotor-Status condition. Correlations were also separated by Group making a total of eight correlations

## 2.4 Apparatus and Materials

*The Spatial Cueing Task* - The spatial cueing task (for a visual depiction, see Fig. 1) used in this study was a modified version of Posner's (1980) paradigm and was run on a PC with an Intel Core i7-6700 CPU and a 27-inch monitor. Each trial began with a fixation stage in which three unfilled, white-bordered boxes (each  $4.5^\circ \times 4.5^\circ$  of visual angle), arranged

horizontally (one in the centre of the screen with the other two  $8.7^\circ$  of visual angle to the left and right of centre) on a black background were presented for 500ms. Following this, a cue would initiate in the form of a randomly selected lateral box thickening from one to 10 pixels for 200ms. An inter-stimulus interval (ISI) then returned the thickened box back to one pixel for 1300ms. After the ISI, a target (a red “X” or “+” each  $2.4^\circ$  in diameter) and a distractor (a white “X” or “+” each  $2.4^\circ$  in diameter) would appear simultaneously, one within the left box and one within the right box. The target stage lasted until a manual response was registered, or at the end of 3000ms with no response. Finally, an inter-trial-interval (ITI) displayed a black background with a single white dot ( $0.4^\circ$  in diameter) in the centre of the screen for a randomised amount of time between 750 and 1250ms, after which, a new trial began.



*Figure 1.* Time course of the spatial cueing task employed in this experiment. Green and orange arrows represent the direction eye position. Eye-tracking dimensions in relation to the display monitor are also included in the bottom left corner.

**EEG** - The EEG system employed in this study was a 32-channel actiChamp gel-based system from Brain Products which sampled at a rate of 256 Hz. Connections between the electrodes and the scalp were achieved using an electrolyte gel (all impedances were kept below 50 mΩ). ERPs were extracted from O1 and O2 electrodes above primary visual sites.

*Manual Responses* - Reaction times were recorded from keystrokes (“z” for leftward targets and “/” for rightward targets) on a Dell PC keyboard.

*Eye-tracking* - Eye-tracking hardware was a desktop mounted EyeLink 1000 Plus from SR Research sampling at 512 Hz to monitor eye position throughout trials.

*ASRS-v1.1 (Kessler et al., 2005; Appendix A)* - The ASRS-v1.1 is a validated questionnaire that assess ADHD symptomology within the general populace. It asks participants to answer on a 5-point Likert scale (never to very often) 18 questions relating to symptoms of ADHD across two sections (attentional and hyperactivity). For each section, answers are summed with scores above 17 indicating that ADHD is likely.

## **2.5 Procedure**

Ethical approval was obtained from the University of Tasmania Human Research Ethics Committee (Appendix B). The experiment took place in light-controlled room where participants were first asked to sit in front of the display computer. After a verbal explanation of the experimental tasks and its apparatus, participants were asked to read an information sheet and sign a consent form (Appendix C and D). Participants were then asked to fill out the ASRS-v1.1 (Kessler et al., 2005) which was linked to experimental data through participant numbers alone, making it non-identifiable. To ensure consistency of visual angle, participants were positioned with their face at a distance approximately 60cm from the computer screen, and their eyeline approximately 5-10cm from the top of the screen. The eye-tracking camera was adjusted so that the participants face would encompass the frame and their right eye was calibrated to the eye-tracking software using a 5-point calibration procedure. After this, an EEG electrode cap was fitted to the participants head. To ensure the cap was correctly centred, measurements of the skull were taken against a reference point on the cap. Anterior to posterior measurements were taken from the bottom of the frontal bone to

the occipital bone and also lateral measurement were taken from the tip of one ear to the other. Electrodes were then attached to the cap.

Before the experiment began, participants were given verbal instructions of how to complete the task. Then, written instructions appeared on the screen immediately prior to task commencement instructing participants on how to perform the task and to which symbol they were to respond. To control for possible response bias for either symbol, twenty participants were assigned to respond to a red “X”, and 20 were assigned to respond to the red “+”. Additionally, to control for order effects, the order in which each Oculomotor-Status condition was administered was counterbalanced. Thus, of the 20 participants who responded to the red “X”, 10 were administered the Om-A level first, and 10 were administered the Om-S level first. The same pattern was applied for the 20 participants that responded to the red “+”.

The cueing task consisted of 24 practice trials to allow the participants to familiarise themselves with the task and then 400 experimental trials – 200 trials for both levels of the Oculomotor-Status condition (100 cued and 100 uncued) which were administered sequentially. Cueing was randomised with cued and uncued trials occurring equally often. Both oculomotor levels of the Oculomotor-Status condition were identical with one exception during the cueing stage: for the Om-A condition, participants were required to make a saccade to the thickened box and then return foveation to the centre box (within 600ms of cue onset) whereas, in the Om-S condition, participants were required to remain foveated on the centre box for the entirety of the cueing stage. With this exception aside, both conditions required participants to remain fixated on the centre box for the entirety of the trial. At target onset, participants were required to respond as fast as they could to the target by performing a corresponding keystroke (“Z” if the target appeared to left and “/” if the target appeared to the right).

If any incorrect saccades were made (e.g., blinks, eyes moved more than  $3^\circ$  of visual angle when they should be fixated, or eyes didn't move to within  $3^\circ$  of visual angle of the correct location when a saccade was required) participants would be shown an error message and the trial was abandoned. Abandoned trials were randomly recycled.

## Results

### 3.2 Behavioural Reaction Times

Results of a 2 (Group: control and deficit) x 2 (Cueing: cued and uncued) x 2 (Oculomotor-Status: Om-A and Om-S) mixed ANOVA revealed a significant main effect of Cueing such that cued trials had slower RTs ( $M = 359.14\text{ms}$ ,  $SD = 46.30\text{ms}$ ) than uncued trials ( $M = 345.20\text{ms}$ ,  $SD = 38.91\text{ms}$ ),  $F(1,31) = 28.24$ ,  $p < .001$ ,  $\eta_p^2 = .48$ . No main effect of Oculomotor-Status was observed for Om-A and Om-S levels returning almost identical RTs,  $F(1,31) = .01$ ,  $p = .947$ ,  $\eta_p^2 = .00$ . There was also no main effect of group with both levels showing very similar overall RTs,  $F(1,31) = .09$ ,  $p = .767$ ,  $\eta_p^2 = .00$  (for further descriptives, refer to Table 1).

While the two-way Group by Cueing interaction was not significant, it approached significance, suggesting that the difference between cued and uncued trials may have differed across levels of Group,  $F(1,31) = 3.16$ ,  $p = .086$ ,  $\eta^2 = .092$ . There was no significant two-way interaction between Cueing and Oculomotor-Status,  $F(1,31) = .01$ ,  $p = .912$ ,  $\eta_p^2 = .00$ , or Group by Oculomotor-Status,  $F(1,31) = .47$ ,  $p = .497$ ,  $\eta_p^2 = .02$ . Additionally, the three-way, group by Cueing by Oculomotor-Status interaction was non-significant,  $F(1, 31) = .01$ ,  $p = .932$ ,  $\eta_p^2 = .00$ .

Given the hypothesis of a Group by Cueing interaction and an observed marginal significance, post-hoc pairwise comparisons were run with Bonferroni corrections ( $\alpha = .025$ ) to further inspect possible differences between cued and uncued trials split between Group levels. Pairwise comparisons revealed a significant effect of Cueing ( $p < .001$ ,  $g = .27$ ) on those in the control level of the Group condition such that cued trials ( $M = 361.68$ ,  $SD = 49.34$ ) were slower than uncued trials ( $M = 345.06$ ,  $SD = 39.39$ ). There was a marginally significant and negligible effect of cueing on those in the deficit level of group showing

slower RTs in cued trials ( $M= 352.91$ ,  $SD= 36.88$ ) than uncued trials ( $M= 344.68$ ,  $SD= 33.81$ ;  $p= .040$ ,  $g= .19$ ).

*Table 1.* Behavioural RT (ms) means, standard deviations, and 95% confidence intervals for the Group by Cueing by Oculomotor-Status analysis.

Group	Cueing		ICEs (Uncued-Cued)
	Cued	Uncued	
Oculomotor	$M (SD)$ [95% CI]	$M (SD)$ [95% CI]	$M (SD)$ [95% CI]
Control ( $n= 22$ )			
Activated	360.28 (49.60) [338.30, 382.27]	343.45(39.16) [326.09, 360.81]	-16.84 (18.32) [-24.50, -9.18]
Suppressed	363.07(52.29) [339.89, 386.25]	346.68(43.80) [327.26, 366.10]	-16.39 (16.10) [-23.10, -9.66]
Collapsed	361.68 (49.34) [341.81, 381.55]	345.07 (39.39) [328.68, 361.45]	-16.61 (14.87) [-22.80, -10.40]
Deficit ( $n= 11$ )			
Activated	354.03 (43.16) [324.75, 383.31]	345.82 (41.64) [321.26, 370.40]	-8.21 (7.58) [-11.40, -5.04]
Suppressed	351.79 (32.24) [323.03, 380.55]	343.54 (28.14) [319.29, 367.79]	-8.25 (8.95) [-12.00, -4.51]
Collapsed	352.91 (36.88) [324.81, 381.01]	344.68 (33.81) [321.51, 367.85]	-8.23 (6.27) [-11.90, -4.52]

### 3.3 P1 Amplitudes

**3.3.1 Ipsilateral electrodes** (for descriptive statistics see Table 2)- Results of an omnibus 2 (Group: control and deficit) x 2 (Cueing: cued and uncued) x 2 (Oculomotor-Status: Om-A and Om-S) mixed ANOVA revealed no main effect of Cueing, indicating similar P1 amplitude modulations across both cued and uncued trials,  $F(1,31)= .03$ ,  $p= .871$ ,  $\eta_p^2= .00$ . There was no main effect of Oculomotor-Status indicating similar P1 amplitudes for

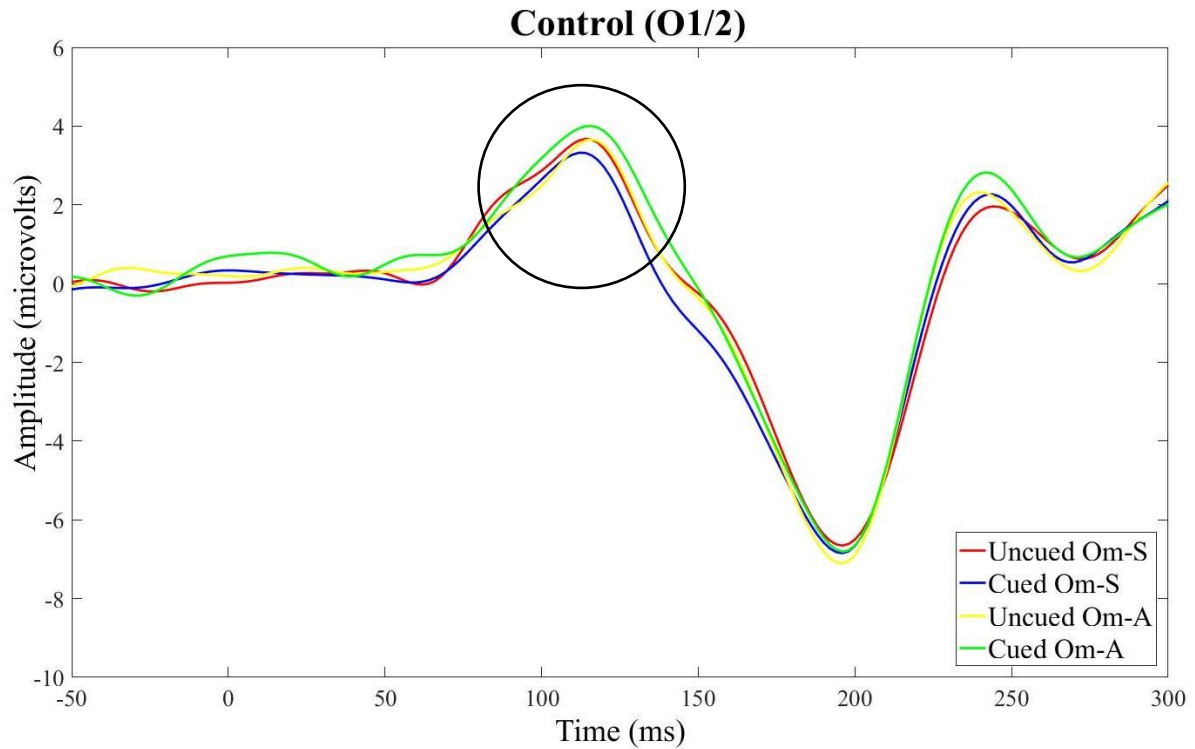


both Om-A and Om-S levels,  $F(1,31)=.71$ ,  $p=.406$ ,  $\eta_p^2=.02$ . There was also no main effect of Group suggesting that amplitude fluctuations did not significantly differ between control and deficit levels,  $F(1,31)=1.76$ ,  $p=.19$ ,  $\eta_p^2=.05$ . (for grand mean waveforms refer to Figures 2 and 3)

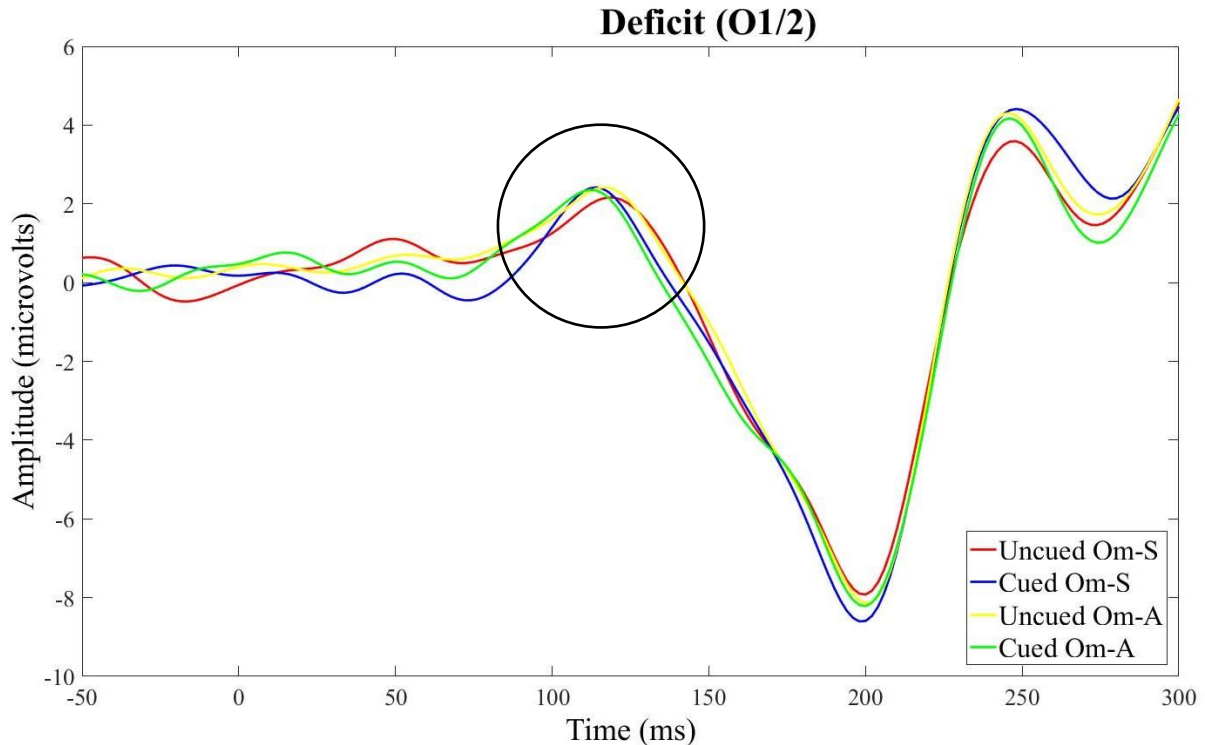
Additionally, the two-way interaction between Oculomotor-Status and Cueing was non-significant indicating that P1 modulations for levels of Cueing did not differ across levels of Oculomotor-Status,  $F(1,31)=1.32$ ,  $p=.260$ ,  $\eta_p^2=.04$ . Also, results showed a non-significant two-way interaction between Oculomotor-Status and Group indicating that P1 modulations for Oculomotor-Status levels did not differ across levels of Group  $F(1,31)=.65$ ,  $p=.425$ ,  $\eta_p^2=.02$ . Non-significance was observed for the Cueing by Group interaction indicating that P1 amplitudes for levels of Cueing remained similar across Group levels  $F(1,31)=.09$ ,  $p=.769$ ,  $\eta_p^2=.00$ . Lastly, the three-way, Group by Cueing by Oculomotor-Status interaction was non-significant,  $F(1, 31)=1.27$ ,  $p=.27$ ,  $\eta_p^2=.04$ .

*Table 2.* Ipsilateral P1 amplitude means, standard deviations, and 95% confidence intervals for the Group by Cueing by Oculomotor-Status analysis.

Group	Cueing		ICE (Uncued-Cued)
	Cued	Uncued	
	<i>M (SD)</i> [95% CI]	<i>M (SD)</i> [95% CI]	<i>M (SD)</i> [95% CI]
Control ( <i>n</i> = 22)			
Oculomotor			
Activated	3.00 (2.36) [1.96, 4.05]	2.58 (2.09) [1.65, 3.50]	.43 (1.16) [-.09, .94]
Suppressed	2.59 (2.12) [1.65, 3.53]	2.97 (2.17) [2.01, 3.93]	-.38(1.40) [-1.00, .24]
Collapsed	2.80 (2.13) [1.85, 3.74]	2.77 (1.89) [1.93, 3.61]	-.02 (1.00) [-.46, .42]
Deficit ( <i>n</i> = 11)			
Oculomotor			
Activated	1.94 (3.60) [-.48, 4.36]	2.02 (3.07) [-.04, 4.08]	-.08 (1.52) [-1.10, .95]
Suppressed	1.44 (2.13) [.01, 2.87]	1.52 (2.04) [.15, 2.00]	-.08 (1.39) [-1.02, .85]
Collapsed	1.69 (2.66) [-.10, 3.48]	1.77 (2.43) [.14, 3.40]	.08 (.81) [-.46, .62]



*Figure 2.* Ipsilateral electrode grand mean waveforms for cued and uncued trials across both levels of Oculomotor-Status for those in the control level of Group. The circled area is the P1 component.



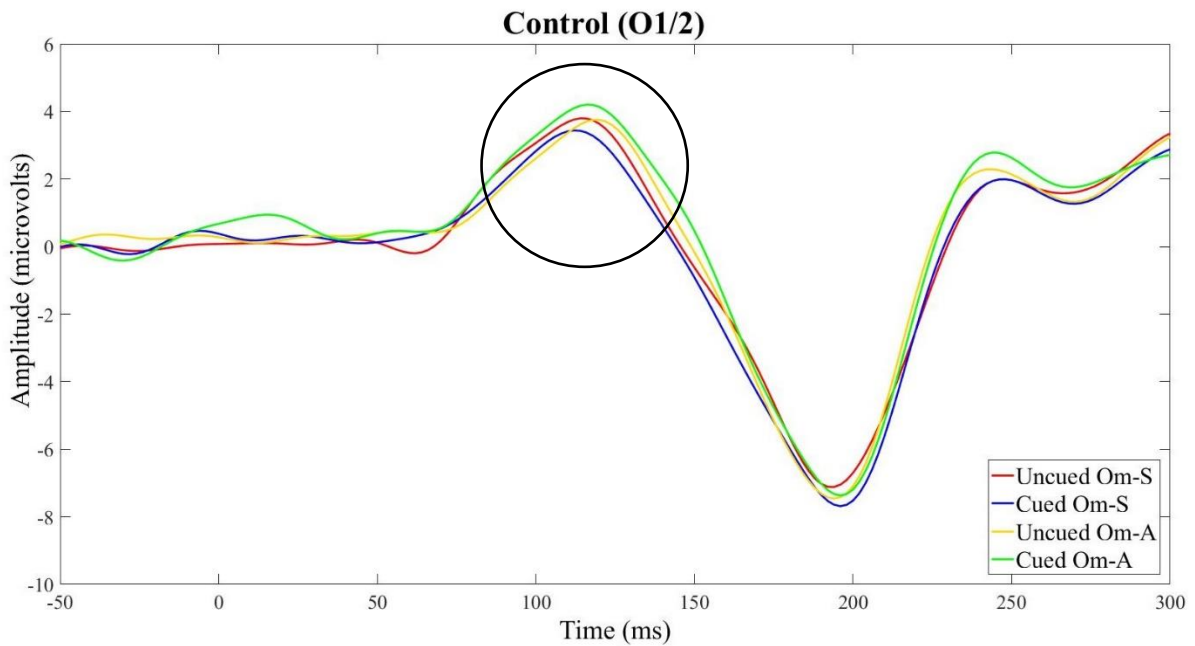
*Figure 3.* Ipsilateral electrode grand mean waveforms for cued and uncued trials across both levels of Oculomotor-Status for those in the deficit level of Group. The circled area is the P1 component.

**3.3.2 Contralateral electrodes** (*for descriptives, refer to Table 3*) - Results of an omnibus 2 (Group: control and deficit) x 2 (Cueing: cued and uncued) x 2 (Oculomotor-Status: Om-A and Om-S) mixed ANOVA looking at P1 amplitudes in contralateral electrodes revealed no main effect of Cueing indicating similar P1 fluctuations across both cued and uncued trials,  $F(1,31) = .26, p = .612, \eta_p^2 = .01$ . There was no main effects of Oculomotor-Status indicating similar P1 amplitudes for both Om-A and Om-S,  $F(1,31) = .78, p = .385, \eta_p^2 = .02$ . There was also no main effect of Group suggesting that amplitude fluctuations did not significantly differ between control and deficit levels,  $F(1,31) = .96, p = .33, \eta_p^2 = .03$ . (for grand mean waveforms refer to Figures 4 and 5)

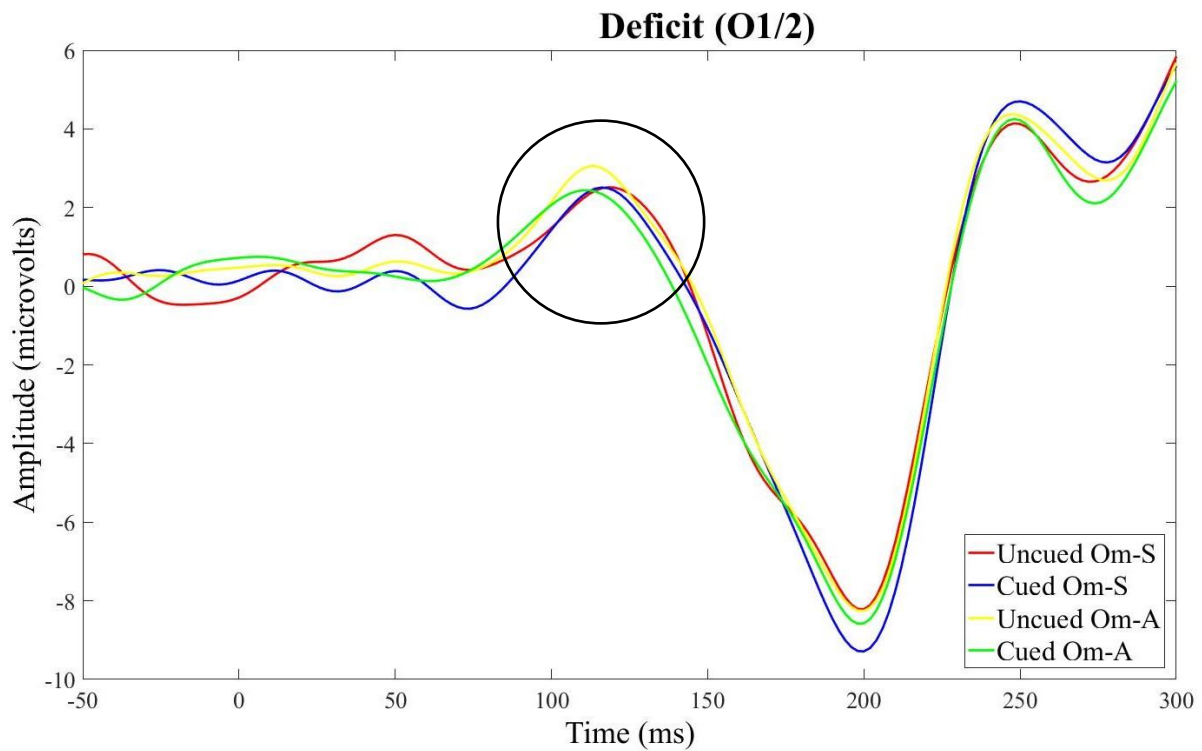
Additionally, the two-way interaction between Oculomotor-Status and Cueing was non-significant indicating that P1 amplitudes for levels of Cueing did not differ across levels of Oculomotor-Status,  $F(1,31) = 1.17, p = .287, \eta_p^2 = .04$ . Also, results showed a non-significant two-way interaction between Oculomotor-Status and Group indicating that P1 amplitude for Oculomotor-Status levels did not differ across levels of Group  $F(1,31) = .54, p = .468, \eta_p^2 = .02$ . Non-significance was observed for the Cueing by Group interaction indicating that P1 amplitudes for levels of Cueing remained similar across Group levels  $F(1,31) = .09, p = .769, \eta_p^2 = .00$ . Lastly, the three-way, Group by Cueing by Oculomotor-Status interaction was non-significant,  $F(1, 31) = 1.84, p = .185, \eta_p^2 = .06$ .

*Table 3.* Contralateral P1 amplitude ( $\mu\text{V}$ ) means, standard deviations, and 95% confidence intervals for the Group by Cueing by Oculomotor-Status analysis.

Group	Cueing		ICE (Uncued-Cued)
	Cued	Uncued	
Oculomotor	<i>M (SD)</i> [95% CI]	<i>M (SD)</i> [95% CI]	<i>M (SD)</i> [95% CI]
Control ( <i>n</i> = 22)			
Activated	3.21(3.75) [1.58, 4.84]	2.75 (3.01) [1.45, 4.05]	.46 (1.54) [-.22, 1.15]
Suppressed	2.75 (2.32) [1.79, 3.71]	3.12 (2.31) [2.17, 4.08]	-.37 (1.38) [-.98, .24]
Collapsed	2.98 (2.91) [1.74, 4.26]	2.94 (2.56) [1.86, 4.02]	-.04 (1.22) [-.59, .50]
Deficit ( <i>n</i> = 11)			
Activated	2.12 (3.75) [-.19, 4.42]	2.43 (2.94) [.59, 4.27]	-.31(1.52) [-1.33, .71]
Suppressed	1.64 (1.98) [.28, 3.00]	1.85 (1.94) [.50, 3.21]	-.22 (1.50) [-1.22, .79]
Collapsed	1.88 (2.74) [.12, 3.64]	2.14 (2.33) [.61, 3.67]	.26 (.98) [-.39, .92]



*Figure 4.* Contralateral electrode grand mean waveforms for cued and uncued trials across both levels of Oculomotor-Status for those in the control level of Group. The circled area is the P1 component.



*Figure 5.* Contralateral electrode grand mean waveforms for cued and uncued trials across both levels of Oculomotor-Status for those in the deficit level of Group. The circled area is the P1 component.

### 3.4 Correlational Analyses

**3.4.1 Oculomotor Activation** - No significant correlations were observed between RT difference and P1 amplitude difference for either control or deficit levels of group, or ipsilateral or contralateral electrodes (for results of correlational analyses see Table 4).

*Table 4.* Correlation coefficients, *P*-values and 95% CIs for correlational analyses in the Om-A level of Oculomotor-Status. Both P1 cueing effects and behavioural ICEs are calculated as a difference score between levels of Cueing (uncued minus cued).

Group	Behavioural ICE		
P1 cueing effect	<i>r</i>	<i>P</i>	95% CIs
Control ( <i>n</i> = 22)			
Ipsilateral	.16	.464	[-.28, .54]
Contralateral	-.19	.406	[-.57, .25]
Deficit ( <i>n</i> = 11)			
Ipsilateral	-.03	.929	[-.61, .58]
Contralateral	-.22	.507	[-.72, .44]

**3.4.2 Oculomotor Suppression** - No significant correlations were observed between RT difference and P1 amplitude difference for either control or deficit levels of group, or ipsilateral or contralateral electrodes (for results of correlational analyses see Table 5).

*Table 5.* Correlation coefficients, *P*-values and 95% CIs for correlational analyses in the Om-A level of Oculomotor-Status. Both P1 cueing effects and behavioural ICEs are calculated as a difference score between levels of Cueing (uncued minus cued).

Group P1 cueing effects	Behavioural ICE		
	<i>r</i>	<i>P</i>	95% CIs
Control ( <i>n</i> = 22)			
Ipsilateral	.06	.807	[-.37, .47]
Contralateral	.08	.716	[-.35, .49]
Deficit ( <i>n</i> = 11)			
Ipsilateral	-.04	.913	[-.62, .57]
Contralateral	.02	.955	[-.59, .61]



## **Discussion**

The primary aim of this study was to explore the modulation of P1 amplitudes modulations and behavioural ICEs in the presence of a distractor stimulus at a long CTOA. As a secondary aim, the present study also wanted to explore the effect that ADHD symptomology in the general populace would have on a study of ICEs.

### **4.2 Behavioural Reaction Time**

Consistent with most of the previous ICE literature (Berlucchi, 2006; Klein, 2000; Samuel & Kat, 2003), the hypothesis that cued trials would show slower RTs than uncued trials for both levels of Oculomotor-Status was confirmed. However, the second part of this hypothesis, that Om-A would have show greater ICEs than Om-S was not confirmed evidenced by a non-significant interaction between Cueing and Oculomotor-Status. Inspection of the means, standard deviations and 95% confidence intervals revealed almost identical variance between cued and uncued trials across both levels of Oculomotor-Status.

These results are at odds with most previous research that find a greater ICE effect in Om-A compared to Om-S (Eng et al., 2018, Hilchey et al., 2014; Satel et al., 2013). Importantly, the present findings are at odds with a recent study (Eng et al., 2018) which found the often-observed Cueing by Oculomotor-Status interaction while employing many of the same attributes as the present study – namely, a distractor stimulus, peripheral cues, and a similar CTOA (1600ms). The only notable difference in their spatial cueing design was that they employed a target and distractor that were both white while the present study used a red target and a white distractor. While the use of a red target seems innocuous, a separate line of research looking at the effects of colour on cognition and behaviour suggests that the colour red may affect processing in ways that could change the observation of ICEs, especially those in the Om-S level. Firstly, exposure to the colour red has been shown to substantially increase

the velocity and strength of motor output (Elliot & Aarts, 2011). It is proposed this is because colour red implicitly activating threat responses. Secondly, in relation to the colour red's relationship with threat response, studies have demonstrated that exposure to threatening stimuli automatically orients attention and activates the oculomotor system (Schmidt, Belopolsky, & Theeuwes, 2012). It is proposed that such responses are caused by a subcortical route to the amygdala (a structure involved in threat response) which passes through the SC (LeDoux, 1996).

Taken together, this evidence suggests that exposure to a red target may be initiating a threat response and implicitly priming the oculomotor system. Thus, the Om-S level, may have experienced implicit priming of the oculomotor system and not actually been suppressed. If this is the case it would make the present studies Om-S level analogous to Rafal et al.'s (1989) planned saccade condition (described in Section 1.2.1), which showed equal magnitude ICEs to their executed saccade condition. That is, while Rafal et al. (1989) used an explicit instruction for participants to plan a saccade which was proposed to prime the oculomotor system, the present study possibly primed the oculomotor system through the activation of a threat response (Schmidt et al., 2012).

The hypothesis that there would be a main effect of Group such that the deficit level would have slower overall RTs than the control level was not supported. This is inconsistent with previous research (Fillmore et al., 2009). However, the second part of the hypothesis, that there would be an interaction between Group and Cueing such that those in the deficit level would have decreased ICEs compared to those in the control level, was partially supported, trending in the direction of previous research (Fillmore et al., 2009). Marginal significance was observed for the interaction suggesting the possibility that ADHD symptomology within a general populace affects ICEs when compared to a control sample. Inspection of the mean difference scores shows that the deficit level's overall ICE was half

that of the control level's. Follow-up pairwise comparisons indicated that those in the control level had a significant ICE, but for those in the deficit level the observed ICE was only marginally significant once appropriate familywise error adjustments were made. However, both had comparably small effect sizes ( $g = .27$  for controls, and  $g = .19$  for deficit) with overlapping 95% CIs, thus, at this point it should be said that this is a trend in the hypothesised direction.

It is interesting that while the present study did not observe the usual overall disparity in RTs between deficit and control, there was a trend toward a reduction in ICEs for the deficit level. One possibility is that overall RT disparities and ICEs disparities are caused differentially across input and output ICEs. For example, Fillmore et al. (2009) showed both overall RT and ICE disparities for ADHD, employing only Om-S. Conversely, Li et al. (2002), showed a trend in decreased ICEs and more comparable overall RTs between deficit and control levels employing only Om-A. This is consistent with the proposal that the present study was not successful in suppressing the oculomotor system in the Om-S level.

### **4.3 P1 Amplitudes**

The hypothesis that a P1 cueing effect would be observed in both Om-A and Om-S was not supported, evidenced by a non-significant main effect of Cueing. Moreover, the non-significant interaction between Cueing and Oculomotor-Status suggests the non-significance in Cueing remained true across both levels of Oculomotor-Status. The hypothesis that there would be an interaction between Group and Cueing was also not supported. No such interaction was observed, indicating that ADHD symptomology had no differential effect on Cueing compared to the control level. Moreover, the results were very similar across electrodes ipsilateral and contralateral to the target.

Overall this indicates that the P1 cueing effect that is observed in most other ICE studies employing EEG was not observed here. One possible explanation of this is the addition of the distractor stimulus that was proposed to partially balance sensory stimulation. Within a single trial RPS is still present on cued trials, however, it is possible that the effect the distractor had across trials was cancelling out this effect. That is, in a standard spatial cueing task, 50% of possible location are being stimulated in any one trial (i.e leftward or rightward cue, and leftward or rightward target). However, with the addition of a distractor, 75% of possible location are being stimulated in anyone trial which increases the likelihood of RPS on uncued trials from one trial to the next. Thus, the absence of a P1 cueing effect in the presence of a distractor is possible evidence in support of the P1 indexing RPS at a long CTOA. These results are inconsistent with both sensory gain control and habituation as the expectation was that the P1 cueing effect should still be observed in the presence of a distractor for both lines of reasoning. For sensory gain control, the effect should be totally reliant on inhibiting the location of the cue (Hillyard et al. 1998) which this study did not change in relation to previous studies. In terms of habituation, because of non-predictive cues, non-associative learning should be dampening sensory response due to the learnt irrelevance of the cue in relation to locating the target (Dukewich, 2009). However, like previous studies, the present study employed non-predictive cues, thus the P1 should still have been observed.

Interestingly, in the absence of a P1 cueing effect altogether, the present study still demonstrated behavioural ICEs. This suggests that the P1 cueing effect is not obligatory when observing input or output ICEs. However, given the rationale for why a behavioural RT Cueing by Oculomotor interaction was not observed, it is possible the present study's Om-S level was not actually suppressed and thus, did not produce input ICEs. Given previous evidence suggesting the P1 is most closely related to input ICEs (Satel et al., 2012; Satel et

al., 2013; Satel et al., 2014), it would be expected that the absence of a P1 cueing effect would be observed in the absence of an input ICE. Thus, in line with the proposal that this study did not successfully suppress the oculomotor system, the relationship between P1 and input behavioural ICEs was potentially not observed. However, while the present study potentially did not achieve true oculomotor suppression, it is still notable that the P1 cueing effect was not observed, as it was hypothesised to occur in the Om-A condition also. Thus, because ICEs were observed in the Om-A condition, this provides further evidence in support of the proposal that the P1 cueing effect is not indexing output ICEs.

#### **4.4 P1 Cueing Effect by Behavioural ICE Correlations**

The hypothesis that the P1 cueing effect would be negatively correlated with behavioural ICEs in the Om-S level but not the Om-A level was not supported. For all correlations calculated, no significance was observed. This is unsurprising given that the present study did not observe a P1 cueing effect. It should also be noted that, if true oculomotor suppression was not achieved in the Om-S level and, instead, output ICEs were observed, then even with a P1 cueing effect, a significant correlation would not be expected, since the P1 cueing effect is thought to index input ICEs. Thus, these findings are potentially still in line with Satel et al. (2013) who demonstrated the dissociation in correlations between Om-A and Om-S levels.

#### **4.5 Limitations**

As has already been mentioned in this discussion, it is proposed that the use of a red target may have implicitly primed the oculomotor system. This potentially confounded the

Om-S level and, therefore, it limited the ability of the study to draw conclusions about ICEs generated during an oculomotor suppressed state. Unfortunately, given the relative obscurity of research on the effects of colour in relation to ICE research, this potential confound was difficult to foresee. With the use eye-tracking to monitor eye position, the present study went to great lengths to ensure the validity of each level of the Oculomotor-Status condition. However, if the proposal made here regarding the effect of using a red target is true, it implies that controls beyond ensuring that eyes remain fixated are necessary to achieve true oculomotor suppression.

The present study was also limited by the design of the EEG caps that were employed. The provided caps did not include electrode sites for PO7 and PO8 areas. Most ICE study's employing EEG take P1 amplitude measurements from PO7 and PO8 electrodes (Satel et al. 2012; Satel et al. 2013; Satel et al., 2014). This is an area more lateral and anterior to the primary visual sites of O1 and O2 (employed in this study) and more in line with where the P1 is proposed to arise (i.e., the lateral occipital complex; Di Russo et al., 2003). While the visual P1 can still be observed at O1 and O2 sites, PO7 and PO8 sites, had they been available, should have produced more pronounced and clearer signal (Luck, Woodmn, & Vogel, 2000). It is possible that, through the present study's use of O1 and O2 sites, important variation was lost through greater diffusion of signal across the scalp.

Another limitation can be seen in the between subjects sample size. While more than enough participants were analysed to satisfy the a-priory power analysis, the assignment of participants to each level of group was restricted by how they responded to the ASRS-v1.1. Only those that scored 17 or above on the ASRS-v1.1 could be considered as part of the deficit level of Group. This meant that far more of the total sample was assigned to the control level ( $n= 22$ ) compared to the deficit level ( $n= 11$ ). It is possible that added statistical power provided by a larger number of participants in the deficit level would have increased

the ability of the study to provide a more definitive conclusion regarding group differences could be drawn.

#### **4.6 Future Research**

Firstly, it should be noted that, at this stage, the proposal that the use of a red target is causing oculomotor priming is speculative. While it is consistent with research on threat response (Schmidt et al., 2012) and processing of the colour red (Elliot & Aarts, 2011) and, in turn, consistent with Rafal et al.'s (1989) findings on the priming of the oculomotor system, research of this nature has not been conducted in the context ICEs. Thus, a possible future direction would be to employ a similar design as the present study with an added 'target colour' condition with at least two levels - a red target level and a more neutral colour (e.g., white). If the proposal made in the present study is correct, a three-way Cueing by Oculomotor-Status by Target-Colour interaction should be observed. The red level (as observed here) should demonstrate a non-significant interaction between Cueing and Oculomotor-Status such that ICEs are the same when executing a saccade and not executing a saccade (due to oculomotor priming). However, this interaction should be significant (as observed usually) for the more neutral colour level such that executing a saccade has a larger behavioural ICE than not executing a saccade (due to differential activation states of the oculomotor system).

In regard to the effect of the distractor on the P1 cueing effect, another possible future direction would be to add a distractor-presence condition in which there would be trials where the distractor would appear with the target (present), and trials where it did not (absent). If the proposal that the distractor is causing RPS across both cued and uncued trials is correct, then a significant P1 cueing effect should be observed when the distractor is absent but not when it is present.

Regarding the exploration the effects of ADHD symptomology on ICEs, the first thing to flag is that future research should have a larger participant count than the present study for those classified as demonstrating deficits. Beyond this, while recruiting individuals who are clinically diagnosed with ADHD was outside the scope of the present study, the reduced ICE for the deficit level observed in this study suggest a similar study with a clinical sample would be beneficial. Another important factor that needs to be considered is that ADHD has a high comorbidity rate with many other disorders including, anxiety disorders major depression, substance use disorders (Kessler et al., 2006). It is therefore, important that future studies use a strict screening process to control for this.

#### **4.7 Summary and Conclusion**

This study explored the relationship between P1 modulations and behavioural input and output ICEs at a long CTOA in the presence of a distractor. It also looked at the effect of undiagnosed ADHD symptomology from within the study's participant sample. Using two Oculomotor-Status manipulations – Om-A to observe output ICEs and Om-S to observe input ICEs – the present study successfully induced behavioural ICEs for both. However, surprisingly, the magnitude of ICEs was the same for both levels of Oculomotor-Status. It was proposed that this was due to the red target symbol implicitly priming the oculomotor system during Om-S and producing output ICEs. Additionally, while there was no overall difference between RTs for deficit and control levels of Group, the deficit level showed decreased ICEs in comparison to the control level. The often observed P1 cueing effect was not demonstrated here which was attributed to sensory balance created by the distractor stimulus. Because no P1 cueing effects were observed, it was unsurprising that there were also no significant correlations between P1 cueing effects and behavioural ICEs.



These results provide further evidence to suggest that the P1 cueing effect is not indexing output ICEs. However, the proposal that the oculomotor system was implicitly primed by the red target limits the present studies ability to draw conclusions about the P1 cueing effects role in input ICEs. It also provides preliminary evidence that ADHD symptomology within the general populace can decrease ICEs. Future studies that manipulate target colour and distractor presence are recommended to respectively explore the effect of colour on ICEs and to further explore the role of sensory adaptation on P1 cueing effects at long CTOAs.

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## Appendix A

A copy of the adult ADHD Self-Report Scale that participants filled out prior to the

### Adult Self-Report Scale (ASRS) Symptom Checklist

experiment. Total scores of 17 or above on either part A or B were classified as a deficit

Participant Number	Today's Date					
Please answer the questions below, rating yourself on each of the criteria shown using the scale on the right side of the page. As you answer each question, circle the correct number that best describes how you have felt and conducted yourself over the past 6 months. Please give this completed checklist to your healthcare professional to discuss during today's appointment.	Never	Rarely	Sometimes	Often	Very Often	Score
1. How often do you make careless mistakes when you have to work on a boring or difficult project?	0	1	2	3	4	
2. How often do you have difficulty keeping your attention when you are doing boring or repetitive work?	0	1	2	3	4	
3. How often do you have difficulty concentrating on what people say to you, even when they are speaking to you directly?	0	1	2	3	4	
4. How often do you have trouble wrapping up the final details of a project, once the challenging parts have been done?	0	1	2	3	4	
5. How often do you have difficulty getting things in order when you have to do a task that requires organization?	0	1	2	3	4	
6. When you have a task that requires a lot of thought, how often do you avoid or delay getting started?	0	1	2	3	4	
7. How often do you misplace or have difficulty finding things at home or at work?	0	1	2	3	4	
8. How often are you distracted by activity or noise around you?	0	1	2	3	4	
9. How often do you have problems remembering appointments or obligations?	0	1	2	3	4	
Part A – Total						
10. How often do you fidget or squirm with your hands or feet when you have to sit down for a long time?	0	1	2	3	4	
11. How often do you leave your seat in meetings or other situations in which you are expected to remain seated?	0	1	2	3	4	

12. How often do you feel restless or fidgety?	0	1	2	3	4	
13. How often do you have difficulty unwinding and relaxing when you have time to yourself?	0	1	2	3	4	
14. How often do you feel overly active and compelled to do things, like you were driven by a motor?	0	1	2	3	4	
15. How often do you find yourself talking too much when you are in social situations?	0	1	2	3	4	
16. When you're in a conversation, how often do you find yourself finishing the sentences of the people you are talking to, before they can finish them themselves?	0	1	2	3	4	
17. How often do you have difficulty waiting your turn in situations when turn taking is required?	0	1	2	3	4	
18. How often do you interrupt others when they are busy?	0	1	2	3	4	

**Part B – Total**

## Appendix B

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26 September 2017

Dr Jason Satel  
Psychology  
University of Tasmania

*Sent via email*

Dear Dr Satel

Re: MINIMAL RISK ETHICS APPLICATION APPROVAL  
Ethics Ref: **H0016857 - Investigating neural mechanisms of visual attention with eye tracking technology**

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We are pleased to advise that acting on a mandate from the Tasmania Social Sciences HREC, the Chair of the committee considered and approved the above project on 26 September 2017.

This approval constitutes ethical clearance by the Tasmania Social Sciences Human Research Ethics Committee. The decision and authority to commence the associated research may be dependent on factors beyond the remit of the ethics review process. For example, your research may need ethics clearance from other organisations or review by your research governance coordinator or Head of Department. It is your responsibility to find out if the approval of other bodies or authorities is required. It is recommended that the proposed research should not commence until you have satisfied these requirements.

Please note that this approval is for four years and is conditional upon receipt of an annual Progress Report. Ethics approval for this project will lapse if a Progress Report is not submitted.

The following conditions apply to this approval. Failure to abide by these conditions may result in suspension or discontinuation of approval.

1. It is the responsibility of the Chief Investigator to ensure that all investigators are aware of the terms of approval, to ensure the project is conducted as approved by the Ethics Committee, and to notify the Committee if any investigators are added to, or cease involvement with, the project.

2. Complaints: If any complaints are received or ethical issues arise during the course of the project, investigators should advise the Executive Officer of the Ethics Committee on 03 6226 7479 or [human.ethics@utas.edu.au](mailto:human.ethics@utas.edu.au).
3. Incidents or adverse effects: Investigators should notify the Ethics Committee immediately of any serious or unexpected adverse effects on participants or unforeseen events affecting the ethical acceptability of the project.
4. Amendments to Project: Modifications to the project must not proceed until approval is obtained from the Ethics Committee. Please submit an Amendment Form (available on our website) to notify the Ethics Committee of the proposed modifications.
5. Annual Report: Continued approval for this project is dependent on the submission of a Progress Report by the anniversary date of your approval. You will be sent a courtesy reminder closer to this date. **Failure to submit a Progress Report will mean that ethics approval for this project will lapse.**
6. Final Report: A Final Report and a copy of any published material arising from the project, either in full or abstract, must be provided at the end of the project.

Yours sincerely

Katherine Shaw  
Executive Officer  
Tasmania Social Sciences HREC

## Appendix C

Faculty of Health, University of Tasmania

Information Sheet (H0016857)



### Investigating neural mechanisms of visual attention with eye tracking technology

#### 1. Invitation

You are invited to participate in a research study looking at how the brain implements mechanisms of visual attention. This study is being conducted by Jason Satel, School of Medicine (Psychology), UTAS.

#### 2. What is the purpose of this study?

The aim of the proposed study is to investigate how different mechanisms in the brain interact when we are looking at a visual scene. For example, if there is a bright flash in front of you, you often can't help but look at it immediately. However, if the same object keeps flashing over and over, you will adapt and stop paying attention to it. We are interested in how [these sort of effects](#) actually work in the brain?

#### 3. Why have I been invited to participate?

You are eligible to participate in this study if you are over the age of 18 and have no existing uncorrected visual disabilities or psychiatric/neurological disorders. Corrected vision through the use of glasses or contact lenses still makes you eligible to participate. All participation is voluntary and there are no consequences either personally or academically if you do not wish to participate.

#### 4. What will I be asked to do?

You will be asked to conduct a series of eye movements and manual responses while completing a computerized task. Your eye movements will be tracked throughout the experiment and your brain activity and reaction times will be recorded. The experimental session should last around 90 [minutes, and](#) will take place in room N121 at the Newnham campus.

#### 5. Are there any possible benefits from participation in this study?

Although there are no direct potential benefits participants or the wider community the study aims to gather knowledge into neural mechanisms underlying visual attention. As compensation for participation, participants will be offered the choice of course credit (1 point/hour) or a dollar value for their time (\$15/hour).

#### 6. Are there any possible risks from participation in this study?

You may experience fatigue over the time of the experiment, if so you may inform the researcher of your discomfort and a break can be scheduled where possible. You may also experience mild skin irritation from the application of conductive gel used during electrode setup if your skin is particularly sensitive.

#### 7. What if I change my mind during or after the study?

You are free to withdraw at any time where there is no obligation to complete participation and no explanation is needed if you choose to withdraw.

#### 8. What will happen to the information when this study is over?

All data collected during this experiment will be confidential and will be destroyed after 5 years.

#### 9. How will the results of the study be published?

At the end of the study, results will be published in academic journals. You can access such articles through the UTAS academic websites.

#### 10. What if I have questions about this study?

This study has been approved by the Tasmanian Social Sciences Human Research Ethics Committee. If you have concerns or complaints about the conduct of this study, please contact the Executive Officer of the HREC (Tasmania) Network on +61 3 6226 6254 or email [human.ethics@utas.edu.au](mailto:human.ethics@utas.edu.au). The Executive Officer is the person nominated to receive complaints from research participants. Please quote ethics reference number H0016857. The individual researcher can be contacted via email at [jason.satel@utas.edu.au](mailto:jason.satel@utas.edu.au).

## Appendix D

### Investigating neural mechanisms of visual attention with eye tracking technology

1. I agree to take part in the research study named above.
2. I have read and understood the Information Sheet for this study.
3. The nature and possible effects of the study have been explained to me.
4. I understand that the study involves paying attention and looking at or ignoring visual stimuli on a computer screen.
5. I understand that participation involves no foreseeable risks, other than the possibility of mild skin irritation during the application of electrodes.
6. I understand that all research data will be securely stored on the University of Tasmania premises for five years from the publication of the study results, and will then be destroyed.
7. Any questions that I have asked have been answered to my satisfaction.
8. I understand that the researcher(s) will maintain confidentiality and that any information I supply to the researcher(s) will be used only for the purposes of the research.
9. I understand that the results of the study will be published so that I cannot be identified as a participant.
10. I understand that my participation is voluntary and that I may withdraw at any time without any effect.
11. I understand that I will not be able to withdraw my data after completing the experiment as it has been collected anonymously.

Participant's name: \_\_\_\_\_

Participant's signature: \_\_\_\_\_

Date: \_\_\_\_\_

#### Statement by Investigator

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I have explained the project and the implications of participation in it to this volunteer and I believe that the consent is informed and that he/she understands the implications of participation.

Investigator's name: \_\_\_\_\_

Investigator's signature: \_\_\_\_\_

Date: \_\_\_\_\_